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1 March 1963 to 30 Msy 1963

INVESTIGATION OF PARAMETERS ESSENTIAL FOR MANNED SPACE FLIGHT OPERATIONS 4 8.49 4 9 W. Move

1. Effect of Elevated Levels of Carbon Dioxide in the Spacecraft Atmosphere.

The purpose of this research was to obtain more definitive data relative to prolonged exposure to ambient $P_{\rm CO2}$ levels of 23 mm Hg at two different barometric pressures. The initial phases of this research were completed prior to receipt of this PR and a portion of the results was presented at the 1963 annual meeting of the Aerospace Medical Association. A copy of this paper is included for your perusal. Analysis of electrolyte studies in regard to this research is in progress and the data will be forwarded to you when it is available.

2. Effect of Ventilating Air Flow on Water Requirements.

Equipment is being ordered and designed for this area of research. The test device should be assembled during the next quarter and check-out should begin shortly thereafter.

3. Oxygen Toxicity Studies on the Hormonal and Metabolic Mechanisms.

Literature pertinent to the effect of high oxygen partial pressure on enzyme systems is being reviewed. Work has started to develop certain enzyme procedures in our laboratories and additional equipment ordered.

4. Examination of Respiratory Requirements and Total Water Balance in a Sealed Environment.

Work has continued on the mounting, instrumentation and checkout of the molecular sieve and its associated vacuum equipment. A CO₂ collection system has been devised and is being checked out. As soon as a suitable chamber becomes available, this will be installed and experimental data obtained.

5. Trace Contaminant Area Gas Chromatography.

Attempts are being made to improve the existing devices by increasing their sensitivity to CO. This is being done by developing a Karmantype ionization detector for on-hand chromatographs. Chambers have been designed to be used as mixing devices for the preparation of standard mixes for IR spectra determination.

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6. Development of a Reliable Gas Analyzing System for Oxygen and Carbon Dioxide.

A bread-board prototype of a non-dispersive IR device for $\rm CO_2$ analyses has been fabricated. This device utilizes no moving parts. At present, full-scale deflection has been achieved at approximately ten percent $\rm CO_2$. The next step is to achieve full-scale deflection with five percent $\rm CO_2$ and to stabilize the baseline.

1 Atch
Paper entitled "Human
Response to Carbon Dioxide
in the Low-Pressure, OxygenRich Atmosphere

HUMAN RESPONSE TO CARBON DIOVIDE IN THE LOW-PRESSURE, OXYGEN-RICH ATMOSPHERE

Captain R. G. Cutler, MC, USAF*
Captain W. G. Robertson, USAF**
Captain J. E. Herlocher, MC, USAF**
F. Ulvedal, Ph.D.**
Captain J. J. Hargreaves, USAF**
B. E. Welch, Ph.D.*

INTRODUCTION

The advent of prolonged manned space missions and the necessary utilization of the scaled atmosphere have increased the requirement for knowledge of man's response to the various anvironmental factors comprising his atmosphere. One such factor, the carbon dioxide concentration in the spacecraft atmosphere, requires further study and definition of tolerance limits, even though the present design limits established for nominal operation (5-7 mm Hg PCO₂) are well within human tolerance. The utilization of CO₂ removing systems which operate more efficiently at higher CO₂ concentrations, the possibility of mechanical failure of the CO₂ removal system, and the probability of increased CO₂ levels resulting from overloads on the CO₂ removal system during rendezvous flights and emergencies, all exemplify the need for accurate, thorough knowledge of human tolerance limits for CO₂ in various spacecraft atmospheres.

Tolerance to chronic exposure to $C\hat{o}_2$ in the sca level atmosphere has

^{*} Chief, Sealed Environment Section, Environmental Systems Branch,
Bioastronautics Department, USAF School of Aerospace Medicine, Brooks AFB, Tex.

^{**} Sealed Environment Section, Environmental Systems Branch, Bioastronautics Department, USAF School of Aerospace Medicine, Brooks AFB, Texas.

Chief, Environmental Systems Branch, Bioastronautics Department, USAF School of Aerospace Medicine, Brooks AFE, Texas.

been the subject of a number of studies and has been discussed by Schaefer 14 and reviewed by others. 2,4,5,17 Reports of alterations of CO2 tolerance at altitude, where hypoxic hyperventilation is superimposed upon the effects of CO2, have also been published; 1 however, the studies are not applicable to the low pressure 0,-enriched artificial atmosphere. Studies by some investigators might lead one to believe that CO2 tolerance would vary with gas density or atmospheric pressure. Lamphier reports elevated alveolar PCO2 levels (PACO2) in working subjects, breathing air under four atmospheres of pressure, but no elevation of PACO, if subjects breathed He2-02 mixtures at that pressure, the latter atmospheres being less dense. On the other hand unjublished studies by our own group had indicated reduction of PACO, in the low-pressure, 02-enriched atmosphere. Such alterations in PACO, with becometric pressure, possibly due to ventilatory changes related to gos dancity, could influence tolerance for CO2. Moreover, studies by Otis and Bomboner 3 demonstrated decreased work of breathing at reduced barometric prossures. Other alterations in pulmonary mechanics such as changes in breath holding time 12 and vital capacity 7,9 at reduced pressures, also raise questions as to the effect of reduced pressure upon tolerance for CO2. The present study was undertaken to obtain additional data regarding man's response to elevated CO2 concentrations particularly comparing responses at 700 mm Hg to those at 200 mm Hg total pressure.

MATERIALS AND METERODS

<u>Fenerimental Sequence</u>. The eight subjects were healthy, young Air Force pilots, who volunteered for the studies. They were carefully coreened prior to

the experiments at the USAF School of Aerospace Medicine Consultation Service for medical and psychiatric ebnormalities.

The eight subjects were divided into groups of four. The first four were carried through the experimental sequence in late 1962 and will be noted by number as 21, 22, 23 and 24, and the second four in early 1963, noted as 25, 26, 27 and 28 throughout the remainder of the paper. The 1962 and the 1963 experiments will be noted as 62-6 and 63-1 respectively.

In daily medical evaluation a survey of symptoms was made by an attending physician. At intervals a physician entered, by means of a lock, into the chamber and personally examined the subjects. Daily routine of the subjects in the chamber was uniform, with a five-hour sleep period, supplemented by two-hour rest periods. Activity was assentially limited to two-hour shifts on a psychomotor testing panel, reading, study, hygiene, personal care, and maintenance of internal chamber facilities. During this experiment the subjects were fed a standardized diet designed by Scrimshaw 15 for study of aspects to be reported elsewhere.

Caloric intake was metered out at 30-40 kilocalories/kilogram.

These studies were conducted in an altitude chamber at the USAF School of Aerospace Medicine in San Antonio which had been adapted for atmospheric control. The CO₂ was monitored by a Beckman LB-1 infrared CO₂ analyzer. The O₂ was monitored by a Beckman F-3 analyzer. Recalibration was carried out every twelve hours. Readings were taken every 15 minutes and the average readings are as presented in Table I.

The subjects were successively exposed to four gaseous environments whose compositions are presented in Table I. The duration of the exposure was four days in each atmosphere carried out in the same sequence as they are listed in

Table I. However, following the exposure to the $\rm CO_2$ -rich 700 mm Hg atmosphere the subjects were removed from the chamber for four days to permit recovery prior to exposure to 200 mm Hg pressure. $\rm CO_2$ levels were maintained to provide comparable tracheal partial pressures of $\rm CO_2$ ($\rm P_{ICO_2}$ = 21 mm Hg, BTPS). This $\rm P_{ICO_2}$ is equivalent to 3 percent $\rm CO_2$ (ETPS) at sea level.

Inspired partial pressure of oxygen (P_{1O_2}) was held to similar levels in each of the four atmospheres such that results would not be influenced by gross changes in PO_2 , hypoxia or oxygen toxicity. Temperature was self-selected by the subjects. In transition from the CO_2 -low to the CO_2 -rich atmosphere, the CO_2 level was gradually increased over a four hour period. The subjects were not told when or how much the CO_2 levels would be elevated, nor what symptoms may be anticipated.

Respiratory Studies. Basal respiratory studies were performed with the subjects fasting and in the recumbent position, after they had awakened from a five hour rest period.

Inspiratory minute volume $(\mathring{\mathbf{v}}_{\mathbf{I}})$ was measured in experiment 62-6 using a servospirometer made by Custom Engineering and Development Company. In experiment 63-1, $\mathring{\mathbf{v}}_{\mathbf{I}}$ was measured with a Tissot spirometer. Both methods gave comparable results. Subjects breathed from seven to ten minutes for collection of the samples. Expiratory minute volume $(\mathring{\mathbf{v}}_{\mathbf{E}})$ was measured by collecting exhaled air in a Douglas bag during the $\mathring{\mathbf{v}}_{\mathbf{I}}$ collection, and measuring its volume in the Tissot spirometer. From these measurements $\mathring{\mathbf{v}}_{\mathbf{I}}$ and $\mathring{\mathbf{v}}_{\mathbf{E}}$ were calculated for the BTPS state. End tidal air (alveolar air) was collected by the Rahn Sampler technique $\mathring{\mathbf{v}}_{\mathbf{E}}$ with the subject recumbent, immediately following the $\mathring{\mathbf{v}}_{\mathbf{I}}$ and $\mathring{\mathbf{v}}_{\mathbf{E}}$ collections. Samples of alveolar air, mixed expired air, and cabin atmosphere were collected

with syringes lubricated with saturated LiCl and were then analyzed.

In experiment 62-6 the oxygen and carbon dioxide content were determined by the Scholander micro-gas analyzer. ¹⁶ In experiment 63-1, these gas contents were determined on the Beckman E-2 oxygen analyzer and the Liston-Becker-1 infrared carbon dioxide analyzer. Comparable results were obtained in each case.

From these studies further derivations could be calculated. Dead space (V_D) was calculated from the Bohr Equation. CO_2 production (\tilde{V}_{CO_2}) , O_2 consumption (\tilde{V}_{O_2}) , and alveolar ventilation were calculated by equations presented by Rahn. 11

The respiratory studies for the eight subjects were then subjected to a repeated measurement-type of analysis of variance, and the statistical significance of differences occurring in the CO₂-rich atmospheres was computed.

Blood Studies. Venous samples were collected anaerobically nearly every day. Analyses of pH and $P_{\rm CO_2}$ were made using pH and $P_{\rm CO_2}$ electrodes, made by Instrumentation Laboratories, Inc. The pH values were then recalculated to that value corresponding to a $P_{\rm CO_2}$ of 40 mm Hg using the Henderson Hasselbalch equation modified as follows:

$$pH + \Delta pH = pK + log \left[\frac{(HCO_3) + 2.54 (Hb)(\Delta pH)}{.0301 pCO_2} \right]$$

where Δ pH = the change in pH

Hb = millimoles of hemoglobin/liter of blood

2.54 = factor of buffer capacity of hemoglobin (-2.54mM HCO3/mMHb/pH unit)

pK = 6.10

The calculation permits evaluation of acid-base balance irrespective of hyper- or hypo-ventilation. The derived values, of course, must be viewed as approximations.

After four days exposure of the subjects to each artificial atmosphere, arterial samples were obtained by brachial artery puncture. Analysis of P_{0_2} was made without delay by means of Clark electrode and the pH and pCO_2 were measured and treated in the same fashion as for the venous blood. End tidal alveolar samples were drawn at the time of arterial puncture by the Rahn sampler for analysis of alveolar gas. It was thus possible to compute alveolar-arterial oxygen gradients.

RESULTS

Subjective Responses. Though the subjects were not told when CO_2 was added to the atmosphere, their acuity was augmented by talking to each other and they were not oblivious to changes in gas injection patterns on the inside of the chamber necessary to alter the chamber atmospheres. In spite of such clues, one subject (27) thought CO_2 not to be elevated at all at 700 mm Hg. Another subject (25) suspected CO_2 elevation one day previous to its actual introduction at 200 mm Hg.

Awareness of hyperventilation was the most helpful physiologic clue, and seven subjects (21, 22, 23, 24, 25, 26 and 28) noted this symptom, particularly in the first two or three days of each CO₂-rich atmosphere.

Thereafter they became less aware of hyperventilating except for subject 22 who was equally aware of it throughout the four days of CO_2 -rich atmosphere exposure. Some subjects noted hyperventilation only intermittently during certain activities, such as when in bed (21 and 22) or while sitting working at the psychomotor panel in the evenings (23 and 24), or when fatigued (25, 26, 27 and 23). This variable awareness of hyperventilating led subjects to suspect that CO_2 levels were intermittently elevated.

Tingling and numbness of the extremities, symptoms suggestive of excessive hyperventilation resulting in hypocapnia, were seen on one occasion in two subjects (25 and 28) during the first day of CO₂ enrichment at 700 mm Hg. Subject 28 also noted light headedness at the same time.

One subject (22) developed a headache on the first day of exposure to the CO₂-rich atmosphere at 700 mm Hg. Five subjects (21, 22, 25, 27 and 28) developed headaches on the first day of exposure to the low pressure, CO₂-rich atmosphere. Three (21, 22 and 25) of the five had intermittent headaches for the next two days. The headaches were mild and generally lasted for one or two hours. No headaches were reported during the low CO₂ periods.

Inspiratory Minute Volume ($\dot{V}_{\rm I}$). Figure 1 shows the day-to-day averages for the eight subjects. It can be seen that there was no appreciable change from the pre-experimental control values to the 700 mm Hg, low-CO₂ atmosphere. However, upon addition of CO₂, $\dot{V}_{\rm I}$ increased and remained elevated throughout the four day CO₂-rich period with no upward or downward trend. Upon return to San Antonio ambient atmosphere, $\dot{V}_{\rm I}$ returned to baseline values.

Throughout the low-CO $_2$ 200 mm Hg atmosphere, four of the subjects (25, 26, 27 and 28) demonstrated moderate hyperventilation, raising the averages during that period to somewhat higher than control values. Again, upon addition of CO $_2$ to the atmosphere $\mathring{V}_{\rm I}$ increased markedly.

Four-day averages for all eight subjects are tabulated in Table II. It can be seen that although subjects began with moderate hyperventilation in the 200 mm Hg low-CO₂ atmosphere, their $\dot{\mathbf{v}}_{\mathbf{I}}$ increment, on addition of CO₂, was nearly the same as in the 700 mm Hg atmosphere.

Analysis of variance of the $\dot{V}_{\rm I}$ values indicated:

- 1. A highly significant increase in V_I upon addition of CO₂, (P<.001).
- 2. A possibly significant increase in $\dot{V}_{\rm I}$ due to the lower pressure alone, (P < .025).
- 3. No significant changes between the pre- and post-experimental values.

Alveolar Carbon Dioxide (P_{ACO_2}) . Figure 2 shows the day-to-day averages for the eight subjects. Changes in alveolar carbon dioxide corresponded to changes in inspiratory minute volume. P_{ACO_2} increased upon addition of CO_2 and decreased when CO_2 was removed. There were no significant upward or downward trends during the CO_2 exposure periods.

Four-day averages for all eight subjects are tabulated in Table III. Corresponding to the relatively greater ventilation during both phases at 200 nm Hg, P_{ACO_2} was relatively less during both phases at that pressure. However, the P_{CO_2} increment on addition of CO_2 was nearly the same at 200 mm Hg as at 700 mm Hg.

Analysis of variance of the PACO2 values indicated:

- 1. A highly significant increase in $P_{\Lambda CO_2}$ upon addition of CO_2 .
- 2. No significant day-to-day trends within any four-day period.
- 3. A significant decrease in P_{ACO_2} associated with the lower pressure (p ℓ .005).
- 4. Possibly significant decrease in the post-experimental P_{ACO_2} values compared with the pre-experiment P_{ACO_2} values.

Other Respiratory Studies. In Table IV it can be seen that respiratory rate changed only slightly in response to the CO₂-rich atmosphere, whereas tidal volume increased by one-third, accounting for most of the increase seen in inspiratory minute volume. This is consistent with results of other investigators for this level of CO₂. ¹³ Figure 3 shows the daily averages for tidal volume and respiratory rate for all eight subjects. The increase in rate during exposures to carbon dioxide was significant at the .01 level. The concurrent increase in tidal volume was likewise significant.

It can be seen in Figure 4 that average daily alveolar ventilation increased upon addition of $\rm CO_2$ to the atmosphere at 700 mm Hg and at 200 mm Hg. The changes here parallel changes in inspiratory minute volume. However, average alveolar ventilation at 200 mm Hg continued to rise throughout the four days, statistically of borderline significance. This upward trend is not reflected in a corresponding drop in alveolar $\rm P_{\rm CO_2}$, and day-to-day fluctuations of this measurement in any given subject were considerable. Thus the significance of this upward trend is not immediately clear and may be a random variation.

In Figure 4, the daily vital capacity averages are also presented.

Though vital capacity was apparently uninfluenced by carbon dioxide exposure.

a decrease occurred in all eight subjects during the exposure to reduced pressure. The average decrease for all eight subjects for the eight-day period compared to the eight days in the 700 mm Hg atmosphere was 10 percent.

In Figure 5, daily averages of oxygen consumption and carbon dioxide production are presented. From the graphs there is a suggestive, gradual downward trend throughout the experiment, possibly due to progressively more basal conditions. Oxygen consumption was found to be significantly lower during the exposure to the CO₂-rich atmospheres than during the four-day control periods. However, those values for the CO₂-rich atmospheres were not lower than the subsequent measurements taken in the recovery and post-experimental phases. Although values for carbon dioxide production paralleled those of oxygen consumption, the greater day-to-day variations preclude any conclusions of significant changes during the carbon dioxide exposure periods.

Figure 6 shows dead space calculations on subjects in the CO₂-low and CO₂-rich atmospheres. Along with some high and spuriously very high values obtained during some days in the chamber, there is a large number of normal determinations during each phase of the experiment. For the eight subjects considered as a group, there is a suggestive trend to slight expansion of the dead space during exposure to carbon dioxide.

In Table V the daily average alveolar P_{0_2} values are presented only to indicate that at no time did alveolar P_{0_2} differ greatly from normal. The changes in themsleves are not significant but only reflect the changes in atmospheric P_{0_2} . Hypoxia was not a factor in this experiment.

Blood pH. The summary of the pH data for venous blood, recalculated to a $P_{\rm CO_2}$ of 40 mm Hg in each case, is presented in Figure 7. The four-day means of blood pH taken during the exposure to the $\rm CO_2$ -rich atmospheres

at 200 mm Hg and at 700 mm Hg were not significantly different from the preceding control periods. The increase in mean pH on the fourth day of $\rm CO_2$ exposure at 200 nm Hg, compared to the preceding three days, was not statistically significant. A 4 mm Hg increment of alveolar $\rm P_{\rm CO_2}$ would be expected to reduce blood pH by approximately .06 pH units. Recalculating all blood samples to a $\rm P_{\rm CO_2}$ of 40 mm Hg cancels out this depression. However, remal compensations to respiratory acidosis tending to increase the blood pH would not be cancelled out but would be reflected in pH values above the baseline. The failure of the mean pH level to show significant increases indicates that a remal compensation, if it occurred, did not measurably affect blood pH.

Alvaeler-Arterial (A-a) Coveren Gradients. The A-a exygen gradients were calculated in order to evaluate the potential development of atelectasis in the 100 percent exygen atmosphere with and without additional carbon dioxide. Thus, venous-arterial shunting of blood consequent to atelectasis could be detected by increases of the A-a gradient. Though possible development of atelectasis in fighter pilots breathing 100 percent exygen has been raised, none of the subjects in the present series of experiments developed appreciable increases in A-a gradients after four days of 100 percent O2 nor after an additional four days of the O2 - CO2 atmosphere. The data is summarized in Table VI. The diminution of the mean A-a gradient during the last arterial puncture was not statistically significant. No evidence of pulmonary atelectasis was found among these eight subjects even after eight days exposure to the nitrogen-free, exygen atmosphere.

DISCUSSION

Of the numerous effects which the CO2-rich atmosphere may have upon

the body, there are two primary and interrelated changes, namely, hyperventilation and hypercapnia. The hyperventilation will limit the degree of hypercapnia and is itself a consequence of the hypercapnia. The many other consequences such as tissue and blood acidesis, increased blood flow to the brain, and alterations in renal excretion, will be secondary to these two effects. Therefore, we have focused attention upon hyperventilation and hypercapnia in evaluating tolerance for carbon dioxide at 700 mm Hg and 200 mm Hg. These pressures were selected to cover a wide range of pressures. For intermediate barometric pressures such as 258 mm Hg (5 psi) used in recent space flights, the expected changes could be interpolated from our results. Below the total pressure of 200 mm Hg (100 percent exygen), however, additional CO₂ will begin to add to the problem of hypoxia. 8

One needs to keep the notable hyperventilation already present in the 200 mm Hg, low GO₂ atmosphere in mind in comparing the nearly identical increases in inspiratory minute ventilation upon addition of GO₂ at each pressure as seen in Table II. Though not all subjects hyperventilated during the 200 mm Hg, low GO₂ atmosphere, none hypoventilated. This hyperventilation at 200 mm Hg was certainly not due to hypoxia since alveolar PO₂ levels were always above 90 mm Hg. It has been reported that 100 percent O₂ at sea level causes hyperventilation and hypocapnia. However, such studies are not comparable to ours, since the alveolar PO₂ in our experiment was essentially normal during the eight days at 200 mm Hg. Another factor is that the 200 mm Hg atmosphere, being less dense, demands less ventilatory affort. Hence, for an equivalent energy expenditure, the individual would be able to ventilate more though no attempt was made in the present study to quantitate this effect.

The small differences in handling of CO2 in the low pressure atmosphere

are only suggestive, namely, a slightly lesser degree of hypercapnia at the expense of a greater degree of hyperventilation. The experimental data presented here demonstrate that tolerance for $\rm CO_2$ in the low pressure atmosphere is substantially the same as for equivalent levels of $\rm P_{\rm CO_2}$, saturated with $\rm H_2O_2$, 37°C at sea level.

The ability of individuals to subjectively detect this amount of carbon dioxide was not always reliable, especially after prolonged exposure. Hyperventilation, which was their most sensitive clue, was less noticeable after the first two days, or when subjects were active and their minds distracted with other matters. Depth of respiration rather than rate showed the greatest change. The fact that when CO2 was added to the chamber, only one subject developed a headache at 700 mm Hg, whereas five subjects developed headaches at 200 mm Hg, is of interest. If the headaches were due to hypercapnia, one would have expected from the alveolar $P_{\rm CO_2}$ studies that more headaches would occur in the 700 mm Hg atmosphere. The variable, unpredictable nature of this symptom precludes firm conclusions in this small series. Moreover, unmeasured peaks of alveolar $P_{\rm CO_2}$ may have occurred throughout the day which could account for the headaches.

Aside from the immediate respiratory response to the elevated atmospheric CO_2 , there is no evidence in these respiratory studies of a gradual accommodation to CO_2 . Though hyperventilation became less noticeable to the subjects in the third and fourth days of exposure to the CO_2 -rich atmospheres, inspiratory minute volume did not diminish nor did alveolar $\mathrm{P}_{\mathrm{CO}_2}$ increase. One might have expected such changes had metabolic compensations of sufficient magnitude taken place. However, four days may not have been long enough for acclimatization to be complete.

SUMMARY

Eight subjects were successively emposed to 21 mm Hg of carbon dioxide (saturated, 37°C), equivalent to three percent at sea level in an atmosphere of 700 mm Hg total pressure and in an oxygen atmosphere of 200 mm Hg total pressure. The duration of exposure to carbon dioxide was four days in each case. Tolerance for carbon dioxide was nearly the same at the two different pressures as measured by the degree of hyperventilation and hypercaphia produced. There was no evidence from the blood studies of a pH shift due to renal or metabolic compensation during the exposure to carbon dioxide. There was no objective evidence in the respiratory studies of an adaptive acclimatization to carbon dioxide during the four-day emposures. There was no development of significant venous-arterial shunts during the emposure to the oxygen atmosphere. The subjective ability to detect carbon dioxide intthe atmosphere is not always reliable, especially after prolonged emposure.

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TABLE I. SUMMARY OF ENVIRONMENTAL CONDITIONS

	Control		e of Atmospher		CO Ptob
	Control	CO ₂ Rich	San Antonio Ambient	Control Low Pressure	CO ₂ Rich Low Pressure
	1 thru 4		s of Experiment 9 thru 12	<u>t</u> 13 thru 16	17 thru 20
Total barometric pressure (mm Hg)	700	700	750 🛣 10	200	200
pCO (saturated 37°C)	2.5 +	.6 21.3 ±.9)	2.57	20. 9 ⁺ 1.0
pO ₂ (saturated 37°C)	148.5+3	.7 155.1 ±3.	.4	142.1 ⁺ 3.6	127.0 ±3.1
pN ₂ (saturated 37°C)	493 .7 11	1.4 437.2 ⁺ 11	1.0	2.7 [±] 2.3	2.7 +1.4
% Relative Humidity	64	80		43	47
Temperature (°C)	24.8	24.2		22.5	22.2

Averages of readings taken every 15 minutes. Standard deviations follow the average readings

TABLE II. INSPIRATORY MINUTE VOLUME (\dot{V}_{1})

3	LOW CO2	HIGH CO ₂	HIGH CO2 MINUS LOW CO2	% Increase
v _I at 700 mm Hg	6 .3 2*	9 . 9 3 *	3.61	57
V₁ at 200 mm H3	6.81*	10.53	3.72	5 5
Increase due to reduced pressure liters	0.49	0.60	· .	
Percent increase due to reduced pressure	8	6		

^{*} Represents liters/minute, BTPS, and is the average of eight subjects over four days.

TABLE III. ALVEOLAR PCO2 (PACO2)

	Lon ${ m co}_2$	нсн со ₂	HIGH CO ₂ MINUS LON CO ₂	% Increase
PACO2 at 700 mm Hg	40.6*	44.3*	3.7	6
$ extsf{P}_{ extsf{ACO}_2}$ at $200~ extsf{mm}$ Hz	33.6*	43.3	7.47	12
Decrease due to reduced pressure	2.0	1.0		
Percent Decrease due to reduced pressure	۲٦	N		

* Represents mm H3 and is the average of eight subjects over four days.

TABLE IV. TIDAL VOLUME, AND RESPIRATORY RATE

	Low CO ₂	High CO ₂	Increment	% Increase
Tidal Volume at 700 mm Hg	.791	1.058	.267 liters	34%
Tidal Volume at 200 mm Hg	.737	.996	.259 liters	35%
Respiratory rate at 700 mm Hg	9.3	10.6	1.3	14%
Respiratory rate at 200 mm Hg	10.2	11.1	.9	9%

The above figures are averages of eight subjects over each four-day period. Tidal volume is expressed in liters, BTPS. Respiratory rate is expressed in cycles per minute.

TABLE V. ALVEOLAR P_{0_2} , em Hg

i,				Days in	the Cha	mber		
	1	2	3	4	5	6	7	8
700 rm Hg	99.0	99.0	105.2	98.5	128.2	123.4	126.8	124.3
200 mm Hg	110.4	108.6	108.8	107.7	104.1	103.1	105.3	105.0

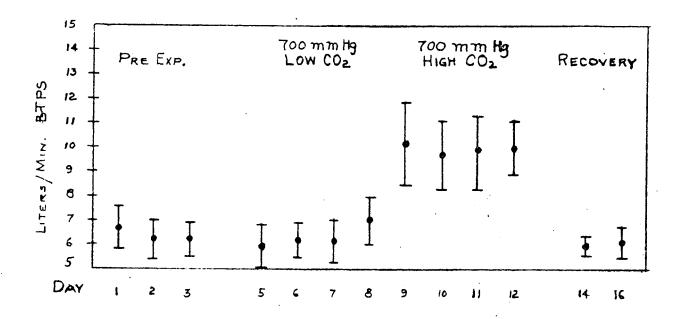
Data presented above are the daily averages of eight subjects. Days 1 to 4 are the low-CO₂ phase and days 5 to 8 are the high-CO₂ phase.

TABLE VI. ALVEOLAR ARTERIAL CRADIENTS

	700 mm Hg low CO ₂	700 mm Hg high CO ₂	200 mm Hg 10w CO ₂	200 mm Hg high CO ₂
Mean PAO2	103.6	123.0	108.4	104.6
Mean PaO2	94.3	114.0	98 .0	100.4
A-a gradient	9.3	9.0	10.4	4.2

Samples were taken after four days exposure to each of the above atmospheres. Each figure represents the averages of 6 to 7 subjects.

Emperiments 62-6 and 63-1



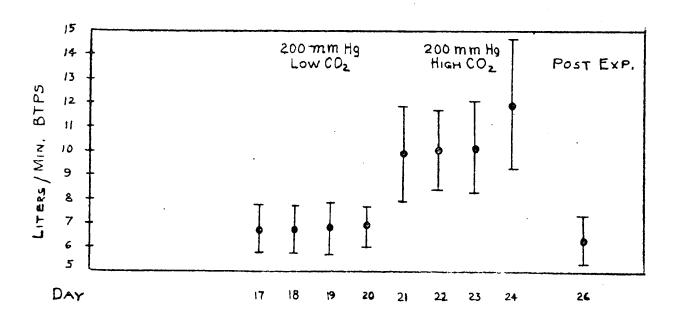
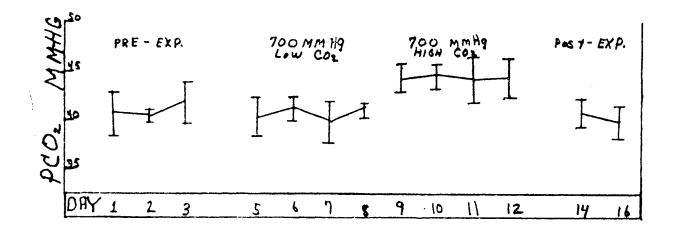


Figure 1. Inspiratory minute volume in liters/minute, BTPS. Daily averages of eight subjects. The lars represent one standard deviation on either side of the mean.



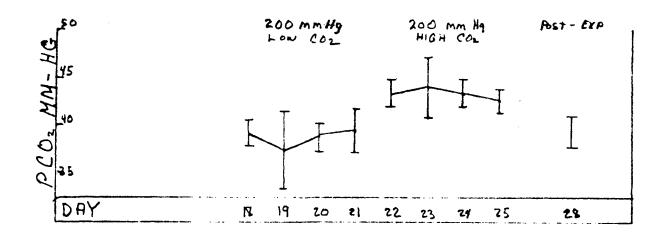
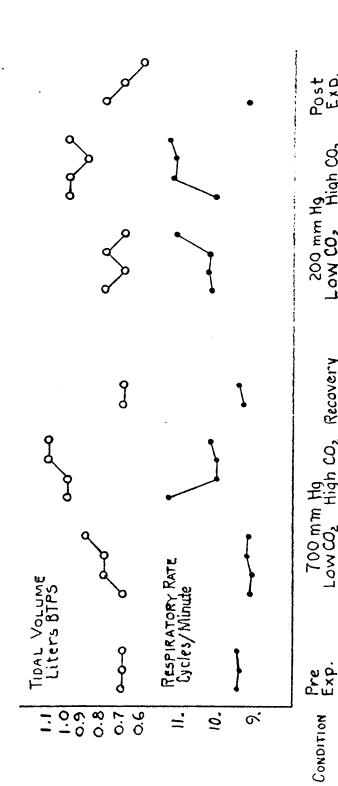
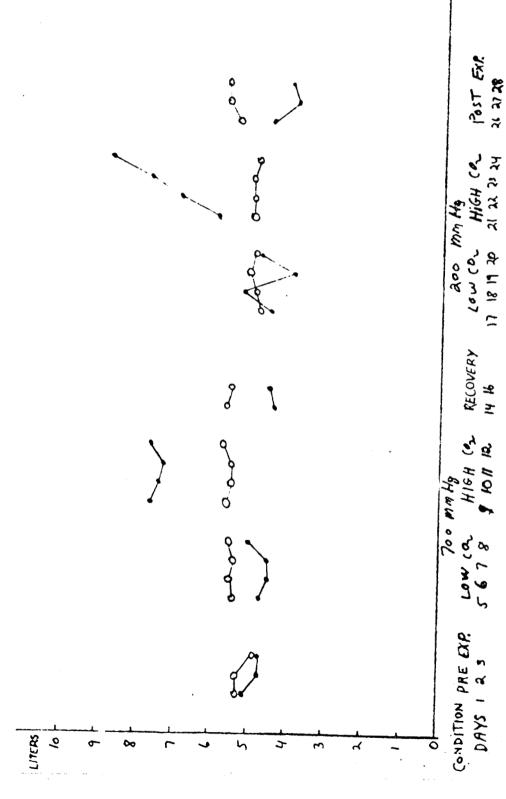


Figure 2. Alveolar $P_{\rm CO_2}$ in mm Hg. Daily averages of eight subjects. The bars represent one standard deviation on either side of the mean.

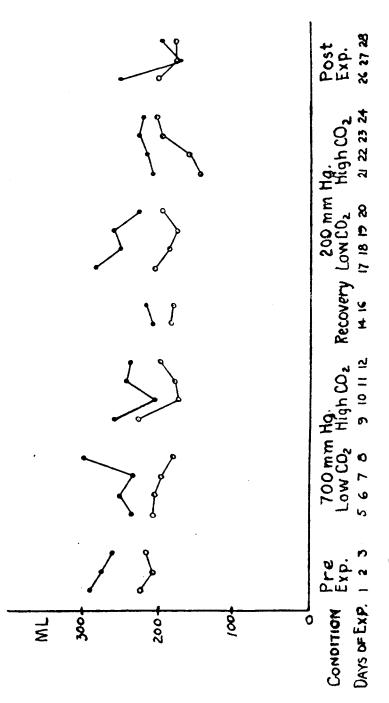


volume is indicated by open circles. Respiratory rate is indicated by closed 26 27 28 Ti dal Post Exp. Tidal volume and reaptratory rate. Daily everages of eight subjects. 21 22 23 24 200 mm Hg Low CO2 High CO2 17 18 19 20 700 mm Hg Low CO2 High CO2 Recovery DAYS OF EXP. 1 2 3 Figure 3.

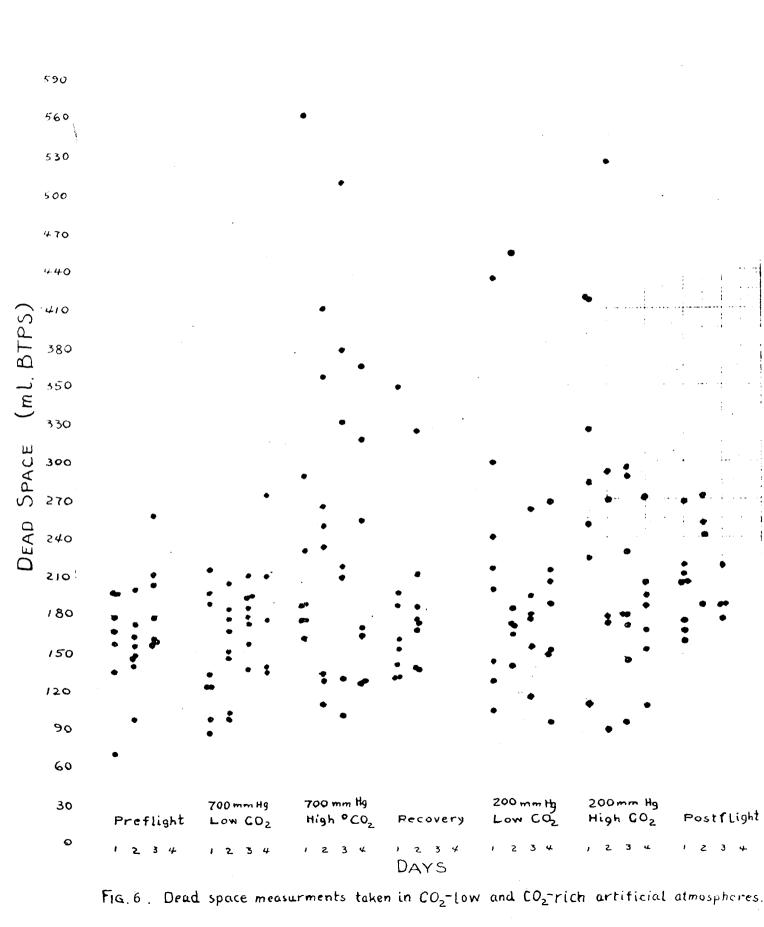
circles.

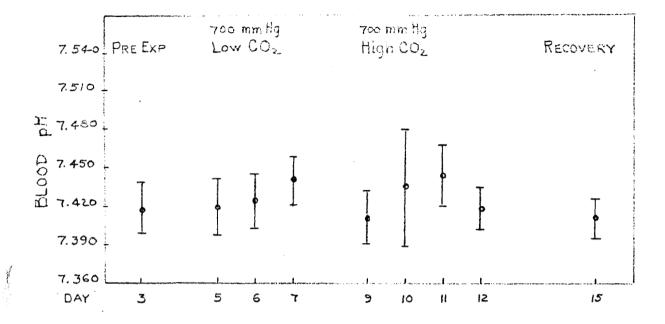


open circles are vital capacity. The closed circles are alveolar ventilation. Vital capacity liters BTPS, and alveolar ventilation determinations in liters/minute BTPS. Averages of eight subjects recorded daily. The Figure 4.



Oxygen consumption and carbon dioxide production in m1/min, STPD, Daily averages of eight subjects, The solid circles represent oxygen consumption. The open circles represent carbon dioxide excretion. Figure 5.





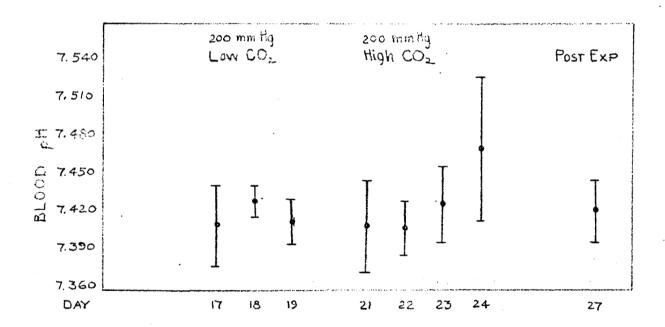


Figure 7. Blood pH. Daily averages of eight subjects. The vertical bars represent one standard deviation either side of the mean.